reveal more cases, especially since we know the groups in which it appears. It is most common in cats and dogs, bovines, predatory birds, parrots, gallinaceous and aquatic birds, and in these groups it is most definitely developed in those living on a protein diet. The lesion is very common in the orders showing the greatest number of cases of gastro-enteritis; this is especially true of parrots, aquatic birds and herbivorous and carnivorous mammals. The aorta is more affected in mammals, the disease is more distributed in birds. A rough similarity to the human aortic lesions is to be seen in the predatory birds and some of the carnivora, while the parrots exhibit degenerations in middle-sized vessels not unlike those of arterio-capillary fibrosis.

The animals most affected are those prepared by nature for severe or prolonged physical effort, such as in fight and flight. Aneurysms occur rarely, usually at points of branching and always near atheromatous plates; they are small and seem to retain part of all coats of the vessels, seldom becoming large enough to form their wall from the surrounding areolar tissue. More males than females are listed in the series, but as we have many more males on record, perhaps the difference would be less marked if the figures were closely comparable.

TWO CASES OF FIBRINOUS BRONCHITIS, WITH A REVIEW OF THE LITERATURE.

By I. Chandler Walker, M.D.,
BOSTON, MASS.

(From the Medical Clinic of the Peter Bent Brigham Hospital, Boston, Mass.)

During the past four and a half years, in which an intensive study on bronchial asthma has been made, the sputum of asthmatic patients has been carefully examined. As a result of careful examination of the sputum 2 cases of fibrinous bronchitis were noted. Although the history which the patient gives, and to a less extent the observation of the patient during an attack suggests the diagnosis, it is the finding of long, branching casts in the sputum that establishes the diagnosis of fibrinous bronchitis.

At various intervals all cases of fibrinous bronchitis, also known as plastic bronchitis and bronchitis psuedomembranosa, have been collected from the literature, but the best, most complete and most recent review of the literature and discussion of the disease was published by Bettman[1] in 1902. Previous to 1869, because of incomplete observations, it is difficult to say just how many cases of fibrinous bronchitis actually occurred in the literature, but a safe

estimation would put the total at about thirty. The interval between 1869 and 1902 covered by Bettman's paper furnishes about 50 cases; for a list of these cases reference may be made to Bettman's article.[1] From 1902 to 1920 I was able to find 31 cases in the literature: 2 of acute fibrinous bronchitis and 29 of the chronic variety. Those cases reported in the literature since Bettman's paper will be listed at the end of this paper.

That the condition is an unusual one is evidenced by the small number of cases reported in the literature; however, the condition is not rare, since the author has learned of local cases that were never reported and of 2 other cases, observed by Dr. Lawrason Brown,[2] that may yet be reported, and, furthermore, while reading the literature, the author ran across several instances in which cases were shown and demonstrated before local foreign medical societies; but since these were never published as cases they are not indexed in the literature on the subject. A more careful examination of the sputum of patients with bronchitis, asthmatic bronchitis and the like would probably result in a still greater yield of cases of fibrinous bronchitis.

Cases of fibrinous bronchitis fall naturally into three groups, namely, acute bronchitis with expectoration of branching casts, chronic bronchitis with expectoration of branching casts and cases in which branching casts were not expectorated but were found in the bronchi at autopsy. Since the chief distinction between the acute and the chronic form is the duration of the disease or the repetition of attacks in the latter, and since most acute cases become chronic because death or permanent relief rarely follows one attack, and since the symptomatology is the same in both types, this paper will discuss only the chronic type of fibrinous bronchitis.

Branching casts may be found in the sputum of diseases other than fibrinous bronchitis. Occasionally in organic heart disease, in pulmonary tuberculosis, in penumonia and frequently in cases secondary to diphtheritic inflammation of the larynx, branching casts are expectorated. Furthermore, it is not uncommon to find small casts without branchings in the sputum of asthmatic bronchitis, and rarely these are found in association with pulmonary edema and lobar pneumonia. None of these conditions, however, are considered to be fibrinous bronchitis, since the formation of casts is secondary or related to some other condition. In other words fibrinous bronchitis is idiopathic, whereas in the condition just mentioned above the formation of casts is symptomatic.

A brief analysis of the symptomatology as usually obtained from cases of fibrinous bronchitis is as follows: The usual age of onset is between the ages of thirty and sixty, with a progressive increase of incidence up to middle life and then a gradual decline. Occupation, family history and chemical irritants seem to have little or no bearing on the disease. Often there is a previous history of some

acute infectious disease, and almost invariably there is a past history of chronic bronchitis. The onset of chronic fibrinous bronchitis is usually with an exacerbation of a chronic catarrhal condition; there may be some fever. In the acute cases there is usually a preceding acute bronchitis, accompanied by considerable elevation of temperature and frequent chills. In both the acute and chronic forms there are paroxysmal attacks of dyspnea and some cough, and these generally immediately precede the expectoration of casts. The dyspnea is chiefly inspiratory in type, the cough is very hard and there is considerable cyanosis and frequently a feeling of tightness or constriction in the chest. Symptoms are temporarily relieved by the expectoration of casts, and the severity of the symptoms are greatly out of proportion to the size or number of casts which, when raised, relieve the attack. Between the raising of large casts, small and incomplete portions of casts are usually expectorated. The amount of respiratory distress would seem to be out of proportion to the degree of limited obstruction caused by the small incomplete casts unless there is a reflex obstruction involving a larger area of the bronchial tree. Hemoptysis is rare. There may be considerable loss of weight and strength.

Physical signs do not distinguish fibrinous bronchitis from ordinary bronchitis, with the exception that when the casts are *in situ* a very coarse, dry, clicking sound, probably caused by the flapping to and fro of loosened portions of casts, may be heard with both inspiration and expiration. Impairment of resonance may be elicited and all types of rales may be heard. One would expect impairment or absence of breath and voice sounds in the area of lung that is obstructed, but this is rarely the case, probably because there is not total obstruction by the casts, which usually have a lumen.

For a detailed description of the casts reference should be made to Bettman's[1] paper. In general the large casts average 10 cm. in length and show branchings down to the seventh degree. They are white in color and have a consistency of fibrin. Many branches have little intumescentia, which may be due to air bubbles or to diverticuli. A lumen may extend throughout the branches, but often the terminal branches end either as a solid plug or as a Curschmann's spiral. Usually small portions of casts are expectorated for some time after a large cast has been expelled. Microscopically the casts usually present a fibrillar, stratified ground substance containing in its meshes leukocytes. The amount of fibrin varies and there is not as much present as one would anticipate. In each case reported only one or two distinct types of bacteria have been found, so that each author has considered that the bacteria found in his particular case were the cause of the condition. On reviewing the literature, however, the bacteria found in the different cases vary so widely that one must conclude either that any type of

organism may cause the condition or that the organisms present in the casts bear no relation to the cause of the disease.

There seem to be no definite sequelæ or complications in this disease, and only rarely is there a fatal termination as a direct result.

CASE REPORT. F. W. P., white, male, aged fifty-five years, was under observation at the Peter Bent Brigham Hospital (Med. No. 22065) from September 22 to September 27, 1919.

The patient complained of cough at night. His parents died of old age and his sisters are well. There is no history of illness or death in the immediate family and no family history of constitutional diseases. The patient has been a farmer all his life and he has always lived in Massachusetts. His past history follows: He had chicken-pox, measles, mumps and whooping-cough in childhood, scarlet fever at the age of six, and six years ago for one year he had rheumatism, swelling and redness of the joints of his fingers and toes, but this did not prevent him from working. He has had a right inguinal hernia for eight years. He has been subject to occasional colds, with some expectoration. Recently he has had nasal obstruction, which was relieved by removal of polypi. He has had no cardiorespiratory, gastro-intestinal, genito-urinary or neuromuscular symptoms. There has been no loss of weight.

The patient's present illness began five months ago, following removal of nasal polypi. Ever since their removal he has had coughing spells of two or three hours' duration almost every night. He is free from these coughing spells during the daytime, and if he sleeps sitting up the cough is much less severe. The paroxysms of coughing are followed by dyspnea of a few minutes' duration. Previous to the cough there is little or no dyspnea, but during the attack both inspiration and expiration are labored. No cyanosis has been noted. At the beginning of an attack he raises frothy sputum, then it becomes slimy, and toward the end of the attack he raises twisted white or yellow strings. There is also a white, watery discharge from the nose during the paroxysm. There is no history of hemoptysis, night-sweats or loss of weight.

Physical examination reveals nothing abnormal as regards the skull, scalp, hair, face, eyes, ears, nose, gums, tongue, pharynx, larynx or neck. He is well developed, thin but not emaciated; his mouth is negative except for a slightly offensive breath, and his teeth are negative, with the exception that there are many fillings and two are missing.

The thoracic cage is small in size and the manubrium is very prominent (chicken-breasted). Chest expansion is good and equal on the two sides; rate and depth of respiration are normal. Examination of lungs reveal no abnormalities, with the exception that a few transient, low-pitched, coarse, bubbling rales are heard, after violent coughing, over the larger bronchi in front on both sides of

the sternum. Examination of the heart, bloodvessels, abdomen, genitalia, rectum, lymphatic glands, bones, skin and extremities reveals nothing abnormal. The systolic blood-pressure is 124 and the diastolic is 80.

Examination of urine, stool and blood revealed nothing abnormal; no eosinophilia. Blood-serum Wassermann reaction was negative.

Roentgen rays showed no abnormality in the sinuses, no pus pockets about the teeth, and the heart to be normal but ptotic in type. The diaphragm was within normal limits; there was diffuse peribronchial thickening throughout both chests; the right apex showed slight mottling and the left hilus showed a slightly increased density.

During the patient's stay of six days in the hospital he slept propped up in bed in order to prevent severe coughing spells. On several occasions, however, about midnight the patient had attacks of difficult breathing, lasting about an hour. Epinephrin injected subcutaneously in 0.5 c.c. doses would give moderate relief; sterile water injected subcutaneously had no effect. The patient's temperature on two occasions was 99°, otherwise it was normal; his pulse ranged from 70 to 90 and respirations varied between 20 and 25 per minute. He had no severe coughing spells while in the hospital, but every day he did raise long branching segments of casts from the smaller bronchi. About ten days previous to entrance to the hospital he had had a severe attack of cough and dyspnea, and at this time he expectorated a large cast.

Examination of the sputum revealed a rather thick, tenacious, slimy material, in which casts and plugs were suspended. The casts were 2 to 5 cm. in length, averaged from 2 to 4 mm. in diameter, had a lumen with various intumescentia and 2 to 4 branches. The plugs were solid molds of the smaller bronchi and some ended in Curschmann's spirals. No tubercle bacilli were found in the sputum; there were a large number of eosinophils present and a few Charcot-Leyden crystals.

Microscopic examination of the casts revealed a fibrillar, stratified, ground substance; the ground substance was mucus and the fibrillar structure was fibrin in the meshes, of which there were polymorphonuclear leukocytes and eosinophils. The solid plugs on section showed a matrix with strings of mucus and fibrin infiltrated with small lymphocytes and polymorphonuclear leukocytes. No tubercle bacilli or influenza bacilla were found either in the casts or in the plugs; many cocci were present.

Bacteriological examination of the sputum alone revealed hemolytic streptococcus subacidus and non-hemolytic streptococcus ignavus; from the casts alone hemolytic streptococcus subacidus and non-hemolytic streptococcus salivarius were recovered. In obtaining these types of bacteria a portion of the sputum was washed and shaken in dextrose bouillon, and then blood-agar plates were poured;

the casts were teased out, washed in sterile saline, shaken and macerated in dextrose bouillon from which blood-agar plates were poured. Smears of the macerated casts and plugs revealed no tubercle bacilli and no influenza bacilli; only cocci in chains were seen.

The second case will not be given in detail, since it has already been published by Dr. Henry A. Christian[3] in the *Medical Clinics of North America*, Boston number, 1919, II, 1255. A summary of this case follows:

D. R., white, male, aged twenty-two years, a plumber's helper by occupation, entered the Peter Bent Brigham Hospital (Medical No. 9990) complaining of breathlessness. His family history, past history, and habits are unimportant. There is no history of exposure to irritating fumes.

The present illness began suddenly three months ago in September, 1918. He was awakened by a severe cough, a feeling of breathlessness and oppression. After an hour the attack gradually passed and he went to sleep. Each night since he has had similar attacks which have increased in frequency, duration and severity. Cough always precedes the attack and the raising of thick, stringy sputum relieves the attack. During the attack expiration is easy, but inspiration is difficult; there is considerable wheezing and cyanosis and a feeling of tightness in his chest. During the day he has mild attacks directly after eating. Epinephrin relieves the attacks. In November, 1919, two months after the first attack, many polypi were removed from his nose without apparent benefit. He has lost forty pounds in weight.

Physical examination is negative, with the exception that examination of the lungs reveals a slightly hyperresonant note throughout; expiration is distinctly prolonged and slightly high pitched and a few scattered crepitant rales are heard throughout the lungs.

Roentgen rays showed that the entire right chest was less radiant than the left and a diffuse peribronchial thickening which is more marked on the right side and dense glands at both lung roots.

The patient was under observation twenty-four days. During this time his temperature usually varied between normal and 99°, with an occasional swing to 100°; his pulse varied between 80 and 100 and his respirations were usually 20 per minute, with an occasional increase to 30 per minute. Every night he had an attack of coughing, dyspnea, chiefly inspiratory, wheezing and expectoration of branching casts. Mild attacks occurred during the day. Epinephrin relieved the attacks. Administration of potassium iodide and an autogenous sputum vaccine seemed to be of no benefit. His blood constantly showed a slight polymorphonuclear leukocytosis.

Repeated examination of the sputum revealed many branching casts, many eosinophils and Gram-positive cocci in chains. No tubercle bacilli were found. A hemolytic and a non-hemolytic

streptococcus in about equal numbers were obtained by plating the sputum in blood agar. Sections of the casts revealed chiefly mucus, with a fibrin reticulum in the meshes of which there were large numbers of mononuclear eosinophils and streptococci.

Discussion of the Etiology of Fibrinous Bronchitis. The etiology of this disease is obscure. In a few cases at postmortem there was a break in the continuity of the bronchial epithelium, in others the bronchial epithelium has contained a large number of mucus-secreting cells which would suggest an exaggerated secretion from the normal mucus glands. The fact that the casts are composed in greater part of mucus would substantiate the increased secretion of mucus and the small amount of fibrin in the casts might come from the rupture or break in the bronchial epithelium. Retention of this mucus and fibrin in the bronchial tree for some time would permit of coagulation or hardening of these substances so that a cast is formed. There would be some irritation causing the accumulation of leukocytes, and since bacteria are normally present in the bronchi, leukocytes and bacteria would naturally be present in the casts.

Since in some cases of fibrinous bronchitis there is an element of neurosis, this condition has been considered by some as allied to mucous colitis. These two conditions are similar in that the intestinal casts of mucous colitis resemble closely the bronchial casts of fibrinous bronchitis, and the etiology of both conditions is obscure.

The author prefers to correlate fibrinous bronchitis with asthmatic bronchitis, bronchiectasis and chronic bronchitis. In almost all of the cases of fibrinous bronchitis, asthmatic bronchitis and bronchiectasis there is a preceding history of bronchitis, and throughout the course of these diseases there is a background of bronchitis. When patients with bronchitis develop paroxysms of dyspnea and suffocation the condition is then called asthma; in order to distinguish this condition from true bronchial asthma the author calls this condition asthmatic bronchitis. For the author's differentiation between true bronchial asthma and atypical or asthmatic bronchitis one may refer to the *Oxford Medicine* by Christian and MacKenzie.[4] If in cases of bronchitis, breaks in the bronchial epithelium, with more or less loss of it occur, fibrinous bronchitis may result; if these breaks invade the deeper structure of the bronchial tubes, thereby causing a weakening of the structure, bronchiectasis may result. In asthmatic bronchitis there is a simple catarrhal desquamation of the bronchial mucus membranes much in excess of that which occurs in bronchitis, and in fibrinous bronchitis there is a permanent loss of the epithelium. Furthermore, in asthmatic bronchitis it is not uncommon to find non-branching casts and plugs in the sputum. Very likely all of these conditions are caused by bacteria, and we know that a large proportion of the cases of bronchitis and asthmatic

bronchitis are due to bacteria, since autogenous sputum vaccines frequently relieve the condition; in fibrinous bronchitis and bronchiectasis, however, vaccines do not benefit, because too much destruction of tissue has taken place. Local lack of vitality probably explains why bacteria produce these conditions in some individuals. Because of the local lack of vitality or inadequate resistance of the bronchi to infection in some persons, bacteria, which in others are non-pathogenic, are able to set up infectious processes. Flurin[5] speaks of the "syndrome de débilité bronchique," which is characterized by hyperesthesia of the mucosa, unstable local circulation and special secretory response to any causes liable to stimulate secretion in the mucous glands.

Conclusions. Cases of fibrinous bronchitis would probably not be as rare as the literature would indicate if the sputa of patients were more carefully examined.

The diagnosis of fibrinous bronchitis is made only by the finding of long, branching bronchial casts in the sputum of cases that do not have tuberculosis, diphtheria, pneumonia or some other primary bronchial disease.

Fibrinous bronchitis is an idiopathic disease, the cause of which is unknown. Although it may be due to a neurosis it seems more closely allied to bronchitis, bronchiectasis and asthmatic bronchitis.

REFERENCES.

1. Bettman, Milton: Report of a Case of Fibrinous Bronchitis, with a Review of All Cases in the Literature, Am. Jour. Med. Sc., 1902, cxxiii, 304.
2. Brewn, Lawrasen: Saranac Lake, N. Y., personal communication.
3. Christian, H. A.: Fibrinous Bronchitis, Medical Clinics of North America, 1919, ii, 1255.
4. The Oxford Medicine. London and New York, Christian and MacKenzie.
5. Flurin, H.: Lessened Resisting Powers of the Bronchi, Paris médical, 1910, ix, 243.

Cases Reported in the Literature Since Bettman's Collection in 1902 up to 1920.

1903 Besc. One case. Montpel. méd., 1903, xvii, 468.
1903 Rabé. One case. Bull. et mém. Soc. anat. de Paris, lxxviii, 708.
1903 Dalché, P., and Hallion, L.: One case. Soc. méd. d. hôp. de Paris, 1903, 3 s., xx, 1101.
1904 Ehrstom: One case. Finska läk. sällsk. handl. Helsingfors, 1904, xlvi, pt. 2, 70.
1904 Rudolph, Wilhelm: Two cases. Schmidt and Klaunig, 1903, Kiel.
1904 Degancllo, U.: One case. Rio veneta di sc. med., Venezia, 1904, xli, 241, 262, 1 pl.
1004 Moser, W.: One case. New York Med. Rec., 1904, lxvi, 216.
1004 Ritchie, W. T.: One case. Edinburg Med. Jour., 1004, n. s., xv, 117–126, 1 pl.
1004 Schwarzkopf, E.: One case. München. med. Wehnschr., 1904, 343.
1904 Giglio, C.: One case. Gaza. d. osp., Milan, 1004, xxvi, 1510.
1904 Menetrier, P.: One case. Pediat. prat., Lille, 1005, iii, 8–10. Ann. de méd. et chir. inf., Paris, 1905, ix, 37–40.
1006 Shoyer, A. F.: One case. British Med. Jour., Londen, 1906, ii, 1035.
1906 Eiselt, R.: One case. Casop. lék. česk, v. Praze, 1906, xlv, 1010.

1007 Dehon, M., and Brasseur, A.: One case, Écho jéd. du nord., Lille, 1907, xi, 80.
1007 Armone, G.: Two cases. Med. ital., Napoli, 1907, v, 381.
1008 Talley, D. D., Jr.: One case. Old Dominion Jour. Med. and S. Richmond, 1098, vii, 06, 1 pl.
1908 West, S.: One case. Lancet, London, 1908, i, 489.
1998 Holmes, E. B.: One case. New York Med. Jour., 1908, lxxxvii, 780.
1908 Schwarz, E.: One case. Wien. med. Wchnschr., 1998, lviii, 1177; 1248; 1307.
1909 Lasnier, A.: One case. Bull. et mém. soc. anat. de Paris, 1999, lxxxiv, 129.
1909 Josué, O., and Paillard, H.: One case. Bull. et mém. soc. de hôp. de Paris, 1909, 3 s xxviii, 97.
1999 Derscheid: One case. Policlin., Brux., 1999, xviii, 101.
1999 Bergman, W.: One case. Wien. u. Leipz., 1998, 342.
1010 von Reuss, A.: One case. Mitt. d. Gesellsch. f. inn. Med. u. Kinderh. in Wien, 1910, ix, 221.
1911 Manec, M. L.: One case. Bordeaux, 1911, A. Saugnac, 99, p. 80.
1911 Griebel, Hermann: One case. Kiel, 1911, H. Fiencke, 21, p. 80.
1915 Israel, Rosenthal: One case. Ugeskr. f. Laeger, Kobenk, 1915, lxxvii, 1412.
1915 Laurio, H., and Colvillo, H. C.: One case. Med. Jour., Australia, Sydney, 1915, i, 145.
1917 Hndley, E. C.: One case. Clin. Jour., London, 1917, lxvi, 297.
1919 Christian, H. A.: One case. Med. Clin. North America, Philadelphia, 1919, ii, 1255.

CURVES OF SUGAR AND UREA AFTER STANDARD PROTEIN MEALS.

By Aage Th. B. Jacobsen, M.D.,
and
Harold Edwards,
new york.

(From the Clinic of Dr. Frederick M. Allen, New York City.)

One of the writers[1] previously reported observations of the effects of diet on the blood sugar of normal persons and diabetics. The results showed no demonstrable influence of protein in the type of cases studied. Rolly and Oppermann[2] observed a rise of blood sugar after protein meals in some diabetics. Strouse, Stein and Wisely[3] found no evidence of a rise of blood sugar in normal persons after carbohydrate-free meals. In animals with suitably severe diabetes controlled by undernutrition, Allen[4] has regularly found marked hyperglycemic curves following protein ingestion. Mosenthal, Clausen and Hiller[5] found that carbohydrate-free meals cause no hyperglycemia in non-diabetics or in some diabetics, but in other diabetics they produce more or less rise of blood sugar. This rise was found particularly in cases with initially low blood sugars, and was commonly missed in cases with marked hyperglycemia at the outset. The rise of blood urea after protein ingestion in rela-

[1] Jacobsen, A. Th. B.: Biochem. Ztschr., 1013, lvi, 471–494.
[2] Biochem. Ztschr., xlix, 1913, 278–292.
[3] Bull. Johns Hopkins Hospital, xxvi, 1915, 211–215.
[4] Am. Jour. Med. Sc., 1917, p. 153, Chart VIII.
[5] Arch. Int. Med., xxi, 1918, 93–108.